UNITED STATES DISTRICT COURT
NORTHERN DISTRICT OF OHIO
EASTERN DIVISION

IN RE: GADOLINIUM-BASED CONTRAST AGENTS PRODUCTS LIABILITY LITIGATION	**Case No. 1:08 GD 50000** **MDL No. 1909** **Judge Dan Aaron Polster**
Paul Decker et al., **- against -** **GE Healthcare, Inc., et al.,**	**Case No. 1:12 GD 50004** **MEMORANDUM OF OPINION AND ORDER**

Before the Court are the following pending motions:

I. Defendant GE Healthcare's ("GEHC") Motion to Preserve and Clarify the Record on Appeal (**Doc #: 106**)

II. Plaintiffs Paul and Karen Decker's Motion to Strike the Supplemental Expert Report of Defense Generic Expert Anthony Gaspari, M.D. (**Doc #: 87**)

III. Plaintiffs' Motion to Exclude Testimony by GEHC Experts as Previously Ruled by this Court (**Doc #: 92**)

IV. Plaintiffs' Motion for Summary Judgment on the Issue of Statute of Limitations **(Doc #: 84)**

V. Defendant's Motion for Partial Summary Judgment on Certain Causes of Action (**Doc#: 88)**

VI. Defendant's Motion to Bifurcate Punitive Damages from Liability at Trial **(Doc#: 105)**

VII. Defendant's Motion for Partial Summary Judgment on Plaintiffs' Design Defect Claim (**Doc #: 89**)

VIII. Plaintiffs' Motion for Partial Summary Judgment on Issues of NSF Diagnosis and Causation (**Doc. #:75**)

IX. Plaintiffs' Motion to Exclude Opinions Relating to Idiopathic or So-Called "Gadolinium Naive" NSF (**Doc #: 81**)

X. Defendant's Motion to Exclude Expert Testimony that "Free" Gadolinium Caused Paul Decker's NSF. (**Doc #: 91**)

The Court will address each motion in the order set forth above, which Roman numerals correspond to the sections below. Because sur-replies are unnecessary in the Court's analysis of the pending motions, the Court **DENIES** the motions for leave to file them. (**Doc ##: 137, 142**.)

I.

On January 11, 2013, GEHC filed a Motion to Preserve and Clarify the Record on Appeal. (**Doc #: 106**.) GEHC asks the Court for an order "expressly confirming the Court's prior decision that all items on the main MDL docket and on the dockets of the four bellwethers (Knase, Bullock, Marino, and Kono) is part of the record in Decker." (Id. at 1.) GEHC also asks the Court to direct the parties "to file any materials relevant to Decker, solely for purposes of the record on appeal, no later than 14 days following entry of judgment after trial." (Id.) Although GEHC seeks to formally preserve all of its prior objections, "this motion does *not* seek to challenge these rulings." (Id. at 1-2 (emphasis in original).) Rather, its purpose is purely procedural, *i.e.*, "to ensure that the record in this case is clear and preserved for any appeal in an efficient manner." (Id. at 2.) Plaintiffs consent to this Motion. Accordingly, the Motion to Preserve and Clarify the Record on Appeal is hereby **GRANTED**.

II.

Plaintiffs ask the Court to strike the supplemental generic expert report of Anthony A. Gaspari, M.D. in its entirety. (**Doc #: 87**.) In 2009, GEHC submitted a generic expert report prepared by Dr. Anthony A. Gaspari, a board-certified dermatologist. (Case No. 1:08 GD 50000 (hereafter, "the Main Case"), Doc #: 736-5 (hereafter, "the Initial Report").) On May 4, 2010, the Court issued a Memorandum of Opinion and Order striking the bulk of Dr. Gaspari's opinions. (Id., Doc #: 788 (hereafter, "Daubert I").) Upon GEHC's request for reconsideration of this ruling, the Court again reviewed Dr. Gaspari's Initial Report, the articles upon which it was based, and his deposition testimony and declined to change its ruling for reasons explained in detail therein. (Id., Doc #: 823 (hereafter, "Daubert II".)

GE recently filed a notice identifying Dr. Gaspari as one of the generic experts it intends to call to testify at the *Decker* trial "consistent with" his initial and supplemental generic expert reports. (Id., Doc #: 94 ¶ 4.) The supplemental report that GEHC has submitted was prepared by Dr. Gaspari on June 30, 2011. (Case No. 1:12 gd 50004 (hereafter, "the Decker case"), Doc #: 87-3 (hereafter "the Supplemental Report".)

After being notified of GEHC's intent to call Dr. Gaspari at trial, Plaintiffs filed the pending Motion to Strike, asking the Court to strike Dr. Gaspari's Supplemental Report in its entirety because, among other things, (1) the report attempts to cure the deficiencies the Court found with respect to his pharmacovigilance opinions and his lack of expertise in that field, (2) his gadolinium-naive opinions rely on the very same studies the Court found methodologically flawed, and (3) he continues to offer opinions about the difficulty diagnosing NSF and other NSF issues which the Court expressly found irrelevant. In response, GEHC

submitted a letter to the Court, asking the Court to strike the pending Motion. (See Decker Case, Doc #: 96, at 1.) The Court denied GEHC's letter request, and directed GEHC to file a brief in opposition to the Motion no later than January 16, 2013. (Id.)

On January 16, 2013, GEHC filed an opposition brief, arguing that the Motion is premature, seeks to broaden the Court's previous rulings, lacks merit, and is nonsensical. (See id., Doc #: 118 (hereafter, "Opp. Br.").)

To save the Court time paraphrasing its prior Daubert rulings regarding Dr. Gaspari, and in the spirit of preserving and bringing clarity to those rulings, the Court will quote those rulings wholesale.

In the 2009 Report, Dr. Gaspari offered two basic opinions – that NSF is difficult to diagnose and that four Adverse Event Reports (AERs) sent to GEHC prior to 2006 were not "clinically" diagnostic of NSF. In Daubert I, the Court excluded those opinions.

> Dr. Gaspari's generic expert report discusses the history of NSF, how it is diagnosed, the differential diagnosis, complicating factors in diagnosing NSF, and an analysis of four Adverse Event Reports received by GEHC. The testimony concerning NSF diagnosis shall not be permitted because it is irrelevant and does not assist the trier of fact. To the extent that GEHC disputes that any of the plaintiffs in the four bellwether trials has NSF, it will need to offer a case-specific expert, who has examined the plaintiff and reached a conclusion on that plaintiff's NSF diagnosis. If there is no challenge to a given plaintiff's NSF diagnosis, generic expert testimony about various NSF diagnostic issues is not relevant. A generic expert testifying at length about how NSF is diagnosed and how other conditions, such as diabetes, complicates NSF diagnosis is not relevant unless the particular plaintiff has diabetes and a case-specific expert testifies that the plaintiff does not have NSF. In that case, the generic expert's testimony would be superfluous.[9]
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> > [AN 9] A case specific expert (such as Dr. Gaspari) who opines that a particular plaintiff does not have NSF could, of course, give the basis for his conclusion, which could include an explanation of

> the difficulty in diagnosing NSF and how other conditions that plaintiff suffers from has complicated the diagnosis.
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> Moreover, Dr. Gaspari may not draw conclusions from the four Adverse Event Reports received by GEHC that GEHC was unaware of Omniscan's potential risks. In his generic expert report, Dr. Gaspari examines four Adverse Event Reports received by GEHC between April 2002 and July 2005 and concludes that the data did not support a diagnosis of NSF in any of these cases and therefore that they did not offer "a compelling, consistent clinical history to alert GE Healthcare to the presence of any association between Omniscan and NFD/NSF." (Doc #: 736-5, at 18.) As observed by Plaintiffs, Dr. Gaspari only examined four Adverse Event Reports and was not provided any preclinical or animal studies conducted by GEHC, any medical literature, or any other relevant data GEHC had at its disposal. Adverse Event Reports examined in a vacuum have significant limitations and are therefore only useful when assessed in the context of other available date. *See* Adverse Event Reports Discussion, Sec. III (B), *supra*. Accordingly, Dr. Gaspari's conclusions are based on incomplete information and therefore do not satisfy the Federal Rule of Civil Evidence 702 requirement that expert testimony be based on sufficient facts or data.

(Daubert I at 53-54.) In short, the Court excluded Dr. Gaspari's pharmacovigilance opinions.

In Daubert I, the Court also precluded Drs. Newton and Waikar from opining that NSF occurs in the absence of gadolinium exposure based on two Deng studies which Dr. Gaspari had co-authored, along with the Wahba and Collidge studies that the Court found similarly flawed. (See Daubert I at 51 (Dr. Newton), at 55 (Dr. Waikar).)

GE subsequently asked the Court to reconsider its rulings, which the Court did in detail. With regard to Dr. Gaspari's pharmacovigilance opinions, the Court affirmed its earlier ruling excluding those opinions:

> The record shows that Dr. Gaspari submitted an expert report, the primary purpose of which was to review the four AERs and "determine whether there was any consistency in [these] reports to suggest that exposures to gadolinium based contrast agents (GBCA) resulted in NFD/NSF." (Doc #: 736-5, at 5.) The report included preliminary information about the history of NSF and the difficulties associated with diagnosing it. This information formed the basis for his conclusion that the AERs did not support a clinical diagnosis of NSF. At deposition, Dr. Gaspari made clear that the purpose for this conclusion was to

> show that the four AERs did not give rise to a "safety signal of NSF." (Id. at 134.) The Court properly excluded this testimony.
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> The question of whether these AERs constituted a safety signal requires someone with *expertise in pharmacovigilance*. The expert must determine whether, given all the information available to GEHC at the time, the AERs gave rise to a safety signal alerting GEHC to the risks associated with administering Omniscan, particularly to the renally impaired.[2] Hence, whether the four AERs supported a clinical diagnosis of NSF is irrelevant to the question of whether the AERs constituted a safety signal.
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> > [AN 2] Dr. Gaspari admitted that the patients in the four AERs all had advanced renal insufficiency. (See, e.g., Doc #: 737-12, Gaspari Dep., at 110.)
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> Dr. Gaspari's deposition testimony makes clear that he is not an expert in *pharmacovigilance*. (See, e.g., Doc #: 737-12, Gaspari Dep., at 81-87.) He repeatedly testified that his sole assignment was to review, *as a dermatologist*, the four AERs and the followup information that was given to him by GE, and to determine whether there were any consistencies or inconsistencies between those AERs, and whether they supported a clinical diagnosis of NSF. (See, e.g., id. at 51, 52, 77, 80, 93, 94.)
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> Furthermore, his report and deposition answers showed that he arrived at his "safety signal" conclusion without reviewing all the information GEHC had available to it at the time, including epidemiological studies (id. at 51-52), relevant GEHC internal documents (id. at 79-80), GE's *in vivo, in vitro* and human studies reflecting GEHC's knowledge regarding gadolinium toxicity (id. at 130-132, 155-163), clinical and preclinical data (id. at 93-94, 145), and the Omniscan label for safety information (id. at 111-115). Rather, he looked only at the information in the AERs and limited followup information given to him by GEHC in opining whether the AERs constituted a safety signal.

(Daubert II at 12-13) (emphasis added).

The Court also affirmed its ruling precluding Drs. Waikar, Newton and Gaspari from testifying that NSF occurs in the absence of GBCA exposure:

> GEHC challenges the Court's ruling precluding Dr. Waikar from testifying that NSF has occurred in the absence of GBCA exposure. The Court properly excluded this testimony because it was based on the Wahba and Collidge studies – the same ones upon which Dr. Newton relied for the same proposition. The Court has again reviewed Dr. Waikar's expert report and finds that, in

addition to the Wahba and Collidge studies, Dr. Waikar cites as an additional basis for this opinion the Deng study. (See Doc #: 677-9, at 37) (citing Deng, A., et al., *Nephrogenic Systemic Fibrosis with a Spectrum of Clinical and Histopathological Presentation: A Disorder of Aberrant Dermal Remodeling*, J CUTAN. PATHOL. Mar 31, 2009.)

This is the same 2009 Deng study the Court previously precluded Dr. Gaspari from relying on to support the identical proposition. (See Doc #: 642, at 2 (prohibiting Dr. Gaspari from opining that NSF occurs in the absence of GBCA exposure based on the Deng article because the authors, one of whom is Dr. Gaspari, stated that possible gadolinium exposure in other hospitals could not be ruled out).) For the same reasons the Court precluded Dr. Newton from relying on the Wahba and Collidge studies for the proposition that NSF occurs in the absence of GBCA exposure, the Court precludes Dr. Waikar from relying on the Deng study for the same proposition.

In reviewing Dr. Gaspari's generic report a second time, the Court observed that Dr. Gaspari states, for the first time in his summary:

> Even in 2009, there is only an association of GBCA exposure, and the development of NFD/NSF in patients with chronic renal failure. There is no clear cause and effect. It is noteworthy that there are *a number of cases* of NFD/NSF occurring in patients with renal failure in the absence of GBCA exposure, *including the case that my group has published* in the clinical dermatology literature (1, 2, 18, 25-35).

(Doc #: 736-5, at 18 (some emphasis added).) The Court notes that, in his deposition testimony, Dr. Gaspari made clear that he was not going to opine on the mechanistic cause of NSF or whether it has been demonstrated that gadolinium causes NSF (Gaspari Dep., Doc #: 737-12, at 55).[3] Yet, he reports that NSF occurs in the absence of GBCA exposure, based now upon fourteen different studies.

> [AN 3] Dr. Gaspari also testified that he was not going to be addressing the history or evolution of NSF. (Id. at 57.)

Interestingly, the first study upon which Dr. Gaspari relies for this opinion is the 2009 Deng study which he co-authored and which the Court earlier precluded him from relying on to support that particular proposition. The second study upon which Dr. Gaspari relies is another article he co-authored that reviews the case of a single NSF patient for whom he was unable to find GBCA exposure – while simultaneously noting that the patient was "lost to clinical followup." (Deng, A., et al., *Localized Nephrogenic fibrosing dermopathy: Aberrant dermal*

> *repairing?* JAAD 2008; 58: 336-9). At deposition, Dr. Gaspari testified that the tissue for this particular patient still exists and has not been tested for the presence of gadolinium. The Court has also reviewed *the remaining twelve articles* upon which Dr. Gaspari relies for the proposition that NSF occurs in the absence of GBCA exposure and finds that *these articles have the same deficiencies as the Collidge, Wahba and Deng studies.*

(Daubert II at 15-17 (emphasis added).)[1] Accordingly, the Court affirmed its ruling precluding Drs. Gaspari, Newton, and Waikar from offering expert testimony on that issue.

The Knase case is a bellwether case that was scheduled for trial on January 24, 2011. (See Knase v. General Electric Co., Case No. 1:08 GD 50026.) In Knase, the Court issued a Memorandum of Opinion and Order on December 6, 2010 that dealt with the plaintiffs' motion to strike the supplemental report of case-specific expert Steven Weisbord, M.D. (Case No. 1:08 GD 50026 (hereafter, "Knase"), Doc #: 257.) Of relevance here, is the Court's ruling denying the motion to exclude *Dr. Weisbord* from testifying that NSF may occur in the absence of gadolinium exposure:

> [T]he Court will not exclude those opinions in the Supplemental Report derived from review of the Lemy study. The Lemy study does not suffer from the same methodological deficiencies as the Collidge and Wahba studies. Unlike the Collidge study, the Lemy authors attempted to determine whether the patient who reportedly developed NSF without exposure to a GBCA may have been administered gadolinium at another facility. The authors of the study noted that, based upon local practice, the transplantation hospital would have to give permission to conduct an imaging scan (with or without a GBCA) on the patient; however, the records indicated that no such permission was granted. (Doc #: 163-2 at 9.) Additionally, in contrast to the Wahba study, the Lemy study conducted a blind test on the tissue of the patient in question using mass spectrometry to confirm the lack of gadolinium in the patients' tissue. (*Id.*)

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[1]For a more detailed explanation of why the Court precluded GEHC's generic experts from opining that NSF occurs in the absence of gadolinium exposure, see Daubert II at 5-8 (Dr. Newton).

> Thus, because it is based on research published after August 2010, which does not appear to contain the methodological flaws of previous studies, the Court will permit *Dr. Weisbord* to opine on the Lemy study. The Court will also grant Plaintiffs leave to supplement expert reports based on the Lemy study.

(Id. at 8 (emphasis added).)

GE has now submitted the Supplemental Report of Dr. Gaspari, and identified him as a generic expert who is expected to provide testimony at the Decker trial that is consistent with his initial and supplemental reports. In the Supplemental Report, Dr. Gaspari *expounds* on his earlier proposition that NSF occurs in the absence of exposure to gadolinium, citing in support of that proposition 19 articles – 14 of which the Court previously found flawed to support that discrete proposition. Not only does Dr. Gaspari *ignore* the Court's previous rulings,[2] he does so with defiance.[3] Indeed, of the 19 studies he cites in support of the proposition that NSF occurs in

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[2]Dr. Gaspari states:

> As discussed in my previous report, there have been similar findings of patients with NSF/NFD who have lacked exposure to GBCAs reported elsewhere in the medical literature. These reports support the conclusion that there are factors other than GBCA exposure that play a role in the pathophysiology of NSF/NFD. (3-15). These articles, as well as my own clinical experience, call into question whether exposure to a GBCA is a necessary co-factor in the development of NSF/NFD.

(Supp. Rpt. at 4.)

[3]Dr. Gaspari states:

> But, Lemy and his co-authors are correct in their description of [patient 5] as a Gd-naive NSF/NFD patient, and that description passed peer-review in a highly respected dermatology journal. This information combined with previously-published articles in the peer-reviewed literature, shows that it would be generally unacceptable and contrary to the scientific method to ignore prior articles reporting NSF/NFD in "Gd-naive" patients.

(Id. at 6.)

the absence of GBCA exposure, the only study that could conceivably support this proposition is the Lemy study.

Because Dr. Gaspari has flagrantly disregarded my previous rulings on this issue, because he cites to and adopts his prior report including *every article the Court specifically and unambiguously excluded as unreliable*, and because the additional articles he cites are similarly flawed to support the proposition that NSF has occurred in the absence of GBCA exposure, his testimony is barred.

With respect to Dr. Gaspari's pharmacovigilance opinion regarding "safety signals," the Supplemental Report shows that, post Daubert I and II, Dr. Gaspari considered what the Court concluded any pharmacovigilance expert should have considered *before rendering an opinion*, i.e., extensive preclinical data, legal documents, clinical trial data, additional AERs, etc. Of course, all of this data was available and accessible to Dr. Gaspari at the time he submitted his Initial Report. More to the point, however, Dr. Gaspari was not then, and is not now, an expert on pharmacovigilance. Accordingly, he is barred from opining on pharmacovigilance matters.

GE contends that Dr. Gaspari's reports disclose, among other things, the history of NSF – a subject they argue may become relevant at trial. But the Court previously ruled, with regard to Dr. Gaspari, that only a case-specific expert who opines that a plaintiff does not have NSF could give the basis for the conclusion, which could include an explanation of the difficulty diagnosing NSF and how other conditions that plaintiff suffers from has complicated that diagnosis. However, GEHC has expressly designated Dr. Gaspari as a generic expert, not a case-specific expert. Accordingly, this argument fails.

GE also argues that the Court's decisions on Dr. Gaspari's generic expert testimony should not be made "before a single witness takes the stand" – and that prematurely ruling on Dr. Gaspari's opinions or expertise today would constitute "an impermissible advisory opinion." (Opp. Br. at 2, 3.) The whole purpose of Daubert proceedings, however, is to determine, prior to trial, whether a challenged expert is qualified to render his opinions and whether he applied the appropriate methodology to support those opinions. Dr. Gaspari cannot cure the many deficiencies in his Initial Report by filing a Supplemental Report citing evidence he should have reviewed before rendering his opinions in the first place, and relying extensively on the very same studies that the Court previously rejected. Nor can supplementing his report turn him into a pharmacovigilance expert when the Court found he was not. Accordingly, the Court **GRANTS** Plaintiffs' Motion to Strike the Supplemental Expert Report of Defense Generic Expert Anthony Gaspari, M.D. (**Doc #: 87**).

III.

Plaintiffs ask the Court to exclude certain testimony from GEHC experts Benjamin Newton, Ph.D., Alan Watson, Ph.D., Steven Weisbord, M.D., and Richard Cohan, M.D. (**Doc #: 92**.) Plaintiffs contend that these experts have submitted supplemental reports that explicitly violate the Court's order restricting such reports to emerging new science or to new studies supplementing old science. Plaintiffs maintain that neither party can ignore the Court's Daubert rulings, or attempt to retroactively cure deficiencies in their 2009 reports with previously available literature or evidence that they failed to consider in the first place.

GE acknowledges that the Court has ruled on multiple occasions that the generic experts could supplement their 2009 opinions with new science. (Doc #: 114, Opp. Br., at 4.) As an

example, GEHC points to the Court's ruling in December 2010 in Knase, denying those plaintiffs' request to exclude Dr. Weisbord from opining that NSF had occurred in the absence of GBCA exposure, based on the Lemy study.[4] (Id.) Apparently, GEHC believes that if the supplemental reports were filed before the deadline for filing them in this case, and the Plaintiffs deposed those experts, Plaintiffs have no basis to challenge those reports or testimony.

The Court has ruled from the get-go that it was not going to allow the parties to relitigate, in Decker, the Daubert rulings the Court had previously made. The Court made clear that what it had formally ruled was admissible – and what it had ruled was not admissible – would still apply with one proviso. If there was emerging new science that somehow modified the generic experts' earlier opinions, those experts would be permitted to file supplemental reports. The Court has never wavered from that representation.

What the Court never intended was the filing of supplemental reports that re-asserted opinions the Court earlier concluded certain experts were not qualified to make. Nor did the Court expect supplemental reports the sole purpose of which was to cure deficiencies the Court previously found in the initial reports. None of that is new science.

With this in mind, the Court is prepared to issue its rulings.

A. Benjamin B. Newton, Ph.D.

In Daubert I, the Court allowed Dr. Newton, a GEHC employee, to opine on flaws with the free gadolinium theory of NSF causation, with particular respect to the findings published in an article he co-authored, Ben B. Newton & Sergio A. Jimenez, *Mechanism of NSF: New*

[4]The Court allowed the testimony because the study was published after the deadline for submitting supplemental expert reports, and because the study did not suffer the same methodological flaws the Court found in the Deng, Wahba, Collidge studies. (See the Knase Case, Doc #: 257, at 5-6; the Main Case, Doc #: 945.)

Evidence Challenging the Prevailing Theory, 30 J.MAGNETIC RESONANCE IMAGING. 1277, 1280-81 (2009). This article sets forth an alternative hypothesis of NSF causation, the primary point being that chelated gadolinium is not as inert as once believed – and that it is chelated gadolinium that stimulates the proliferation of fibroblasts, monocytes and macrophages, cells that are present in the early stages of NSF.

The Court also determined that,

> [a]long with challenging the free gadolinium theory, Dr. Newton may also provide limited expert testimony on whether it was foreseeable that GBCA exposure would lead to the injuries encountered by renally compromised NSF patients. His degree in pharmacology and background as a translational scientist and cellular biologist, which is the basis for his foreseeability opinions, provide him with the necessary expertise to offer a reliable opinion.
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> However, Dr. Newton's lack of significant training or experience in dermatology, pathology or histology makes his testimony challenging the similarity of rat lesions to the symptoms of NSF unreliable. It is clear from Dr. Newton's deposition that he lacks the requisite level of comfort with basic dermatohistology or dermatologic clinical manifestations, such as dermal induration, to render a reliable expert opinion on this topic. Additionally, Dr. Newton may not testify about the relationship between *in vitro* and *in vivo* chemical stability of Omniscan. Dr. Newton's background does not include the focused study of chemistry necessary to be considered an expert on the stability of chemical compounds.

(Daubert I, at 51-52.) Finally, with regard to Dr. Newton's opinion that NSF occurs in the absence of gadolinium exposure, the Court concluded:

> The Court, however, will not permit Dr. Newton to testify that NSF has occurred in the absence of GBCA exposure. The Court agrees with Plaintiffs that the two studies cited by Dr. Newton in support of this proposition - Wahba IM, Simpson EL, White K. *Gadolinium Is Not The Only Trigger For Nephrogenic Systemic Fibrosis: Insights From Two Cases And Review Of The Recent Literature*. 7 AM. J OF TRANS. 1-8 (2007) and Collidge TA, Thomson PC, Mark PB, *et al*. *Gadolinium-Enhanced MR Imaging And Nephrogenic Systemic Fibrosis: Retrospective Study of a Renal Replacement Therapy Cohort*. 245 RADIOLOGY 168-175 (2007) – are fundamentally flawed. The Collidge study did not examine whether the one NSF patient who had not received a GBCA while

> undergoing an MRI had undergone any non-MRI procedures in which a GBCA was used. The Wahba study, which concluded that two patients developed NSF without exposure to a GBCA, did not confirm its findings by testing these patients' tissue for the presence of gadolinium.

(Id. at 53.) In short, the Court closed the door on Dr. Newton opining that NSF occurs in the absence of GBCA exposure based on these particular studies.

The Court has reviewed Dr. Newton's supplemental report. (Doc #: 129-11.) Therein, he cites recent data and development in the field which "bolster[s] the hypothesis that the persistent pathology underlying NSF is triggered by a single high GBCA dose in susceptible patients and that gadolinium found in tissues, e.g. bone and skin, may play a very limited, if any, role in the development of this condition." (Id. at 2.) The Court sees no reason to exclude Dr. Newton's opinions on this issue with one important exception.

Dr. Newton opines that the hypothesis that retained gadolinium causes NSF has become "untenable" because recent studies show, among other things, that "patients with no history of GBCA administration have developed NSF." (Id. at 11.) Further, "[t]here is a growing number of reports of NSF in patients not exposed to GBCA, raising further questions about the role of retained gadolinium in the aetiology of NSF." (Id. at 12.) And, "NSF has been diagnosed in more and more patients who have no history of GBCA administration suggesting that other factors trigger the pathology of NSF." (Id. at 20.) He supports this proposition with articles the Court previously found unreliable to do so, with the sole exception of the Lemy study. As before, Dr. Newton is precluded from opining that NSF may occur in the absence of GBCA exposure based on any study or article but the Lemy study. And if Dr. Newton is unable to opine without discussing the studies the Court has found unreliable to support that proposition, he may not testify about it at all.

B. Alan C. Watson, Ph.D.

Plaintiffs seek to exclude certain testimony by GEHC expert Alan C. Watson, Ph.D. In Daubert I, the Court dealt with the challenge to Dr. Watson's generic expert report and testimony as follows:

> The Court will not permit defense expert Dr. Alan Watson to testify on matters outside the field of bioinorganic chemistry. Dr. Watson holds a B.Sc. degree in Chemistry, a Ph.D. in Coordination and Bioinorganic Chemistry and an M.B.A. Since receiving his M.B.A. in 1988, his responsibilities appear to have been more on the business side than on the science side.
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> Dr. Watson's expert report contains the following substantive subject headings: (1) The Need for GBCAs; (2) GBCA Design and Stability; (3) Thermodynamic and Kinetic Stability Factors; (4) Macrocyclic Chelate Complexes as GBCAs; (5) Toxicity Studies of GBCAs; (6) The Issue of Gadolinium Retention; (7) Skin Lesions During Preclinical Testing; (8) The Early History of Salutar; (9) Salutar and Nycomed Publications; (10) Nycomed's Own Internal Documents; (11) Foreseeability; and (12) Commonality of All the Current Commercially Available GBCAs.
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> Given that Dr. Watson's responsibilities have been primarily on the business side at least since 1999 and perhaps since 1988, he is not qualified to provide expert testimony on most of the subjects discussed in his expert report. Because of his background, Dr. Watson is qualified to speak generally about bioinorganic chemistry, the study of how inorganic elements function in living organisms. The topics which may correspond to his knowledge of bioinorganic chemistry are: The Need for GBCAs, GBCA Design and Stability, Thermodynamic and Kinetic Stability Factors, Macrocyclic Chelate Complexes as GBCAs and The Issue of Gadolinium Retention. Dr. Watson does not have the requisite expertise to opine on the other issues within his report. In particular, Dr. Watson may not provide an opinion on the FDA's review of Omniscan, his assessment of Omniscan or GBCA toxicology or toxicology studies, any preclinical or clinical studies of Omniscan (including those done by Salutar and Nycomed), analysis of relevant publications on GBCAs, or foreseeability of gadolinium poisoning.

(Daubert I at 52-53.) Upon GEHC's request for reconsideration, the Court affirmed its prior ruling:

> GEHC objects to the fact that the Court allowed Drs. Parisian and Fine to interpret outside publications and testify regarding the significance of clinical trials, while precluding Dr. Watson from analyzing similar, or in some cases identical, publications and clinical trials. However, Dr. Watson's expertise is significantly different than Plaintiffs' two experts. As explained *supra*, Dr. Watson is a biochemist, not a medical doctor, and he has not worked directly on the scientific side of the pharmaceutical industry for at least twenty years. He has little to no experience analyzing clinical data, and little experience in any other field outside of biochemistry. Dr. Parisian, on the other hand, is a board-certified anatomic and clinical pathologist with a Masters Degree in Biology. She has particular experience reviewing and evaluating clinical trials through her work at the FDA. Dr. Fine is a board-certified nephrologist and an Associate Professor of Medicine. He has particular and extensive experience diagnosing, researching and treating NSF, and has published numerous articles on this topic since 2003. The Court allowed Drs. Parisian and Fine to analyze clinical trials and relevant publications because they have the experience necessary to understand and opine on them. However, Dr. Watson lacks the requisite expertise to provide a legitimate analysis of documents outside the field of biochemistry. Because Dr. Watson's background and experience are significantly different than the that of Drs. Fine and Parisian, they cannot properly be compared. As such, the Court properly precluded Dr. Watson from testifying about matters outside his field of bioinorganic chemistry.

(Daubert II at 17-18.)

Plaintiffs ask the Court to strike documents and related testimony from Dr. Watson's supplemental report which are based upon documents available to him prior to August 31, 2009, the date he submitted his initial report. Plaintiffs contend that Dr. Watson has not supplemented his initial report with new science. Rather, they contend that he is simply re-addressing Dr. Karen Saebo's relaxometry study and Dr. Jerrold Abraham's free gadolinium studies – both issues upon which the Court found Dr. Watson unqualified to opine.

GE argues that Dr. Watson discusses "new studies that 'provide a clear and convincing alternative explanation for the interpretation of Muller and Saebo's data.'" (Suppl. Rpt. at 3.) According to Dr. Watson, "these studies . . . clearly establish the extent to which protein binding is the correct explanation for the relaxometry results obtain by Muller and Saebo." (Id. at 3.)

According to GE, Dr. Watson offers precisely the kind of supplemental opinions this Court expected and permitted in Knase. (Knase Final Pretrial Hrg. Tr. (Ex. 1) at 57, 59-60.) Moreover, his discussion of Dr. Abraham is a response to Dr. Cramer's deposition and the new studies carried out by Dr. Cramer and Dr. Abraham. (Supp. Rpt. at 1.)

The Court has reviewed Dr. Watson's Supplemental Report. Therein, he expounds at length on the studies conducted by Drs. Saebo and Muller between 1988 and 1993, and on Dr. Abraham's EXAFS studies. (Doc #: 115-1.) Suffice it to say that Dr. Watson may only opine on the fields to which he was limited in 2010 and, based on his own admissions at his 2008 deposition, he is again precluded from opining about areas outside of bioinorganic chemistry – including toxicology, clinical studies, and pharmacology.

C. Steven D. Weisbord, M.D., M.Sc., FASN

On August 31, 2009, Dr. Steven Weisbord prepared an expert report on generic issues common to all MDL cases on behalf of GEHC – which report GEHC filed on March 15, 2010. (See the Main case, Doc #: 736-1.) On September 28, 2010, Dr. Weisbord prepared a Supplemental Report. (Id., Doc #: 933-3.) On November 8, 2012, the Plaintiffs' Steering Committee filed a motion to exclude the supplemental generic expert reports of Drs. Weisbord and Waikar. (Id., Doc #: 933.)

Meanwhile, on July 8, 2010, Dr. Weisbord prepared an expert report on issues related specifically to the Knase plaintiffs. (See the Knase case, Doc #: 136-1.) On September 8, 2010, the Knase plaintiffs filed a motion to exclude or limit the case-specific testimony of Dr. Weisbord. (Id.; Doc #: 136.) And on October 11, 2010, the Knase plaintiffs filed a motion to strike Dr. Weisbord's supplemental generic report. (Id., Doc #: 163.)

On December 6, 2010, the Court issued opinions in the Knase and Main cases regarding the Supplemental Report. (Knase; Doc #: 257; the Main Case, Doc #: 945.) As a preliminary matter, the Court noted that GEHC had moved yet another time for reconsideration of that part of Daubert I related to case studies of NSF victims with no antecedent GBCA administration. (Id. at 2 (citing Doc #: 891).) The Court explained therein that it held a telephonic hearing with counsel on September 15, 2010, during which the Court denied GEHC's motion. The Court noted, however, that if GEHC's experts believed that the science had changed, GEHC could propose a supplemental expert report based upon that new science. (Id. (citing Hrg. Tr. at 5-6.) With regard to other generic issues, the Court held:

> Accordingly, the Court will not consider untimely those aspects of the Supplemental Report addressing new science not available to Dr. Weisbord prior to his August 31, 2009 Expert Report. Exhibit A to the Supplemental Report contains a comprehensive list of additional documents reviewed by Dr. Weisbord. Most of these documents were available to Dr. Weisbord prior to August 31, 2009. Therefore, those parts of Dr. Weisbord's Supplemental Report derived from his review of *documents available prior to August 31, 2009* are stricken and Dr. Weisbord may not testify based upon this information.
>
> Similarly, the Court will not permit Dr. Weisbord to render any opinions in the Supplemental Report based upon the declarations of GEHC employees Aud Moxnes, Carl Einar Sjogren, and Per Trygve Normann, because those declarations are not based on *new science or research published since August 31, 2009*.

(Doc #: 945, at 3.)

Now, as then, the Court will not exclude those opinions in Dr. Weisbord's Supplemental Report that are derived from review of the Lemy study. However, if Dr. Weisbord is unable to opine that NSF may have occurred in the absence of GBCA exposure without discussing the studies the Court has already found flawed, he may not testify about it at all.

D. Richard Cohan, M.D.

Plaintiffs point out that Dr. Richard Cohan has submitted a supplemental report to his generic expert report that includes a list of 49 references—32 of which cannot possibly be considered new science because they date back as far as 1989, 1991 and 2006. (Doc #: 92-1, at 10.) According to Plaintiffs, "[t]he fact that GE failed to supply its own internal documents to its own expert prior to the deadline for generic expert reports in this MDL is not a valid reason for allowing such a supplementation." (Id.).

Despite its best efforts, the Court has been unable to find either Dr. Cohan's initial generic expert report or a ruling excluding any of his opinions in that report. Obviously, Dr. Cohan may not opine that NSF has occurred in the absence of GBCA exposure based on any of the studies this Court has found deficient. Otherwise, the Motion (**Doc #: 92**) is **DENIED** as to Dr. Cohan.

IV.

Plaintiffs have filed a motion for summary judgment, asking the Court to rule that Mr. Decker's claims are not time barred. **(Doc #: 84)**. The parties agree that the applicable statute-of-limitations period is two years. See O.R.C. § 2305.10(A). The parties also agree that the two-year clock did not start running until the earlier of the following dates: when Paul Decker was informed by competent medical authority that his NSF is related to exposure to GBCA; or when he should have known through the exercise of reasonable diligence that his NSF is related to GBCA exposure. *See Id.* at (B)(1). This case was filed on February 2, 2012.

With respect to the first date, there is no dispute: Decker was informed by competent medical authority in August 2010 – well within the two-year limitations period—that he had

NSF as a result of his exposure to gadolinium. As to the second date, there *is* a dispute.

Plaintiffs argue that August 25, 2010, is the earliest date on which Decker could have known he had NSF because it was on that date he received the NSF diagnosis; before then, his doctors believed he had – and formally gave him a diagnosis of – scleroderma. Defendant disagrees, contending that Decker's "treating physicians may have informed him as early as August 2009 – outside the applicable two-year limitations period – that he had NSF associated with his MR procedure with contrast in 2005." (Doc. #: 126 at 9).

Even though Decker was not formally diagnosed with NSF until 2010, the onset of his NSF symptoms began as early as 2006. One of his regular nephrology appointments took place on July 31, 2009. During the appointment, Decker complained of skin tightening. His nephrologist wrote in his office notes: "Differential diagnosis would be systemic sclerosis [scleroderma] versus nephrogenic systemic sclerosis [NSF]." (Doc. #: 84-14). When asked at his deposition whether he had discussed the potential NSF diagnosis with Decker, the nephrologist replied, "I most likely did discuss it." (Doc. #: 126-5 at 3).

In August and September of 2009, Decker was seen by a different nephrologist. During these visits, the doctor considered NSF as a possible cause of Decker's symptoms. When asked at his deposition if he mentioned NSF to Decker, the nephrologist responded, "I would conjecture that I probably did." (Doc. #: 126-10 at 5). Both of these nephrologists asked Decker whether he had received an administration of GBCA with his MR procedure.

These facts give rise to a genuine dispute—at least when viewed in the light most favorable to Defendant. Given that Decker was told by two nephrologists during a span of two months that NSF was a possible diagnosis, that those doctors gave him reason to think his 2005

GBCA administration was medically relevant to his condition, that he had NSF symptoms as early as 2006, the widespread availability of information—both in the medical community and online—about NSF, and the fact that Decker declined his nephrologist's invitation in early 2009 to seek treatment for his skin hardening (specifically, he declined to have his legs tested), a rational juror could conclude that, with the exercise of reasonable diligence, he should have figured out in 2009—outside the applicable limitations period—that his disease is related to his gadolinium exposure. Summary judgment is therefore not appropriate; the issue is for the jury to decide. Accordingly, this motion **(Doc #: 84)** is **DENIED.**

V.

GEHC has filed a two-part motion for partial summary judgment on certain causes of action (**Doc. # 88**). In the first part, GEHC observes that all ten of the Deckers' common-law product-liability claims[5] have been abrogated by the Ohio Products Liability Act. GEHC is correct, and Plaintiffs concede the point. See O.R.C. § 2307.71(B) (declaring that the Act is "intended to abrogate all common law product liability claims or causes of action"). The remedy, the parties acknowledge, is to allow Plaintiffs to amend the complaint, which they must do promptly. The jury instructions, which the parties are to file by February 14, should, of course, track the elements of the Ohio Products Liability Act.

In the second part of its motion, GEHC argues that the Deckers are barred from recovering punitive damages. The resolution of this issue turns mainly on O.R.C. § 2307.80. That statute allows for the recovery of punitive damages where the manufacturer "manifested a

[5]They are: manufacturing defect, design defect, defect due to inadequate warning, defect due to nonconformance with representations, negligence, breach of express warranty, breach of implied warranty, misrepresentation, fraud, and loss of consortium.

flagrant disregard of the safety of persons who might be harmed by the product in question." *Id.* at § 2307.80(A). But the statute has a special rule applicable to claims involving drugs that have received FDA approval.

The rule is that a manufacturer of an FDA-approved drug is not liable for punitive damages unless the manufacturer fraudulently withheld from the FDA material and relevant information or the manufacturer misrepresented such information to the FDA. O.R.C. § 2307.80(C)(1)(a). But there is another obstacle: only the FDA gets to determine whether the manufacturer committed fraud or misrepresentation.

In Garcia v. Wyeth-Ayerst Labs., 385 F.3d 961 (6th Cir. 2004), the Sixth Circuit considered a Michigan statute that authorized a fraud-on-the-FDA tort claim. The question before the Court was whether the statute is preempted by federal law. The Court gave a clear, but conditional, answer. The Court explained that "state tort remedies requiring proof of fraud committed against the FDA" are preempted unless "the *FDA itself* determines that a fraud has been committed on the agency during the regulatory-approval process." Id. at 966 (original emphasis). To allow otherwise – to permit a court to make an independent finding of fraud – would lead to "inter-branch-meddling." Id.

The same goes for the second exception to the general punitive-damages bar – misrepresentation of material and relevant information to the FDA. The Sixth Court, in a published opinion, Marsh v. Genentech, Inc., 693 F.3d 546, 551 (6th Cir. 2012), cited with approval a recent unpublished opinion, In re Aredia & Zometa Prods. Liab. Litig., 352 F. App. 994 (6th Cir. 2009), for the proposition that the rule in Garcia applies to "claims that the manufacturer misrepresented or withheld information about a drug from the FDA after the FDA

had approved it." In short, a punitive-damages claim for an FDA-approved drug is allowed under Ohio law *only if* the FDA has made a finding of either fraud or misrepresentation. There is no such finding here.

Plaintiffs offer a twofold response. They argue that this Court already decided in their favor. Not so. In the relevant opinion, which was an opinion in the Knase case, the Court ruled that the plaintiffs were not precluded on preemption grounds from presenting evidence that GEHC withheld key scientific and safety information from the FDA in support of causes of action *other than* fraud on the FDA, such as a failure-to-warn claim. (Id., Doc. # 263.) Indeed, the plaintiffs in that case were asserting neither a fraud-on-the-FDA claim nor any claim that requires proof of fraud. This case is different. Here, as the Ohio statutory scheme makes clear, Plaintiffs *must* prove fraud on the FDA or misrepresentation to the FDA in order to recover punitive damages – which, of course, they can't do absent an FDA finding of fraud or misrepresentation.

Plaintiffs' second angle of attack is to distinguish the Sixth Circuit cases mentioned above. They argue that in those cases the plaintiffs brought claims for fraud on the FDA, whereas in this case Plaintiffs have brought, not a claim for fraud, but a claim for punitive damages. To clarify, punitive damages are not a stand-alone cause of action; they are a remedy for certain causes of action. But the distinction matters little here, because the Court, in articulating the relevant holding in *Garcia*, used the phrases "cause of action," "claim," and "tort remedies" interchangeably. In one sentence the Court used the phrase "a state common law fraud-on-the-FDA tort *claim*." Garcia, 385 F.3d at 966 (emphasis added). A few sentences later, the Court spoke in terms of a "*cause of action* for fraud on the FDA." Id. (emphasis added).

And at the end of the same paragraph, the Court, in stating its conclusion, wrote that "state tort *remedies* requiring proof of fraud committed against the FDA" are preempted absent an FDA finding. (emphasis added). The point is clear; the *Garcia* rule applies to every legal theory – whether the theory is part of a claim or remedy – that requires proof of fraud on, or misrepresentation to, the FDA. For these reasons, punitive damages are unavailable to Plaintiffs.

VI.

Since punitive damages are no longer part of this case, Defendant's motion to bifurcate punitive damages from liability at trial (**Doc#: 105)** is **DENIED AS MOOT**.

VII.

GEHC has filed a motion for partial summary judgment on Plaintiffs' design-defect claim. (**Doc. #: 89**). GEHC contends that Plaintiffs cannot successfully make out a design-defect claim because they are unable to prove an element: a practical, feasible alternative design that would have prevented the harm. See O.R.C. § 2307.75(F); McGrath v. GMC, 26 Fed. Appx. 506, 510 (6th Cir. 2002). GEHC argues that, because *no* GBCA is safe for renally impaired patients like Mr. Decker, no safe alternative to Omniscan exists.

GEHC bases its argument on the opinions of two radiologists: Mr. Decker's treating radiologist, Dr. Phillip Shaffer; and Plaintiffs' radiology expert, Dr. Richard Semelka. Dr. Shaffer, in his deposition testimony, said he no longer uses GBCAs in his practice on renally impaired patients because the risks outweigh the benefits. GEHC also quotes the testimony of Plaintiffs' radiology expert, Dr. Richard Semelka, who opines that the decision to forego GBCAs altogether in renally impaired patients is consistent with the current standard of care.

But Plaintiffs have ample evidence that there is now – and was well before Mr. Decker received his MRI scan – safe alternatives to Omniscan.

In 1989, Nycomed, GEHC's predecessor company, convened a task force to review the safety of Omniscan and other GBCAs. The company considered two types of GBCAs that are differentiated on the basis of their molecular structure: linear, or single-chain, GBCAs and macrocylcic, or ring-like, GBCAs. Omniscan is an example of a linear contrast agent; Prohance and Dotarem, which are used by the healthcare company Bracco, are of the macrocyclic variety. The task force recognized that macrocyclic structures are safer than linear structures because linear structures are less stable than macrocyclic structures. Because they are less stable, linear GBCAs more easily become untethered to the binding agent, releasing, it is feared, "free" gadolinium that can remain in the body of individuals whose kidneys have trouble filtering it out (*i.e.*, renally impaired patients). The task force concluded that macrocyclic GBCAs are both more effective and safer than linear GBCAs. Other scientists have likewise concluded that a linear structure is more toxic than a macrocyclic structure. (See Doc. # 121-10). Nycomed even developed and tested – with positive results – a macrocyclic GBCA. The reason it never made it to market, Plaintiffs claim, is that the manufacturing costs were an order of magnitude higher than the costs associated with Omniscan.

GEHC responds to this evidence by arguing that, even if there are alternative designs, none of these designs would have *prevented* Mr. Decker's NSF, as the relevant Ohio statute requires. But it is perfectly logical to infer that a macrocyclic GBCA, which is significantly less likely to release "free" gadolinium in a patient's body, would have prevented – or at least slowed the development of – NSF in Mr. Decker. Such an inference is premised on Plaintiffs' free-

gadolinium theory, which, as we shall see, is a valid, relevant theory of what caused Mr. Decker's NSF. Moreover, one of the radiologists that GEHC cites to support its position undermines it with his statement that if Mr. Decker had received Magnevist, rather than Omniscan, he would have been less likely to develop NSF. (See Doc. # 90-2 at 18). GEHC's argument is also called into question by the article "Nephrogenic Systemic Fibrosis/Nephrogenic Fibrosing Dermopathy in Advanced Renal Failure," in which the authors acknowledge that GBCAs may be appropriate in some renally impaired patients, in which case they recommend a macrocyclic, not a linear, GBCA be used. (Doc # 122-5 at 9, 13). The issue is clearly one for the jury. Consequently, the Motion (**Doc #: 89**) is **DENIED**.

VIII.

Plaintiffs have filed a motion for partial summary judgment on the issues of NSF diagnosis and causation. (**Doc #: 75**.) According to Plaintiffs, there is no dispute that Mr. Decker has NSF and that his Omniscan-enhanced MRI caused it. They contend that Mr. Decker's medical records uniformly show that he has NSF, his physicians have uniformly testified that he has NSF, his case-specific experts have opined that he has NSF, and counsel for GEHC has represented that GEHC "will not call a witness to dispute Mr. Decker's NSF diagnosis." (See Motion, Ex. 1, December 3, 2012 C. Tisi Letter to J. Philip Calabrese.) Furthermore, GEHC's case-specific experts, Drs. Sica and Weisbord, will neither dispute that he has NSF nor that the September 2005 Omniscan administration caused his NSF.

More to the point, GEHC has not sought to contradict either Mr. Decker's NSF diagnosis or its cause through either of its two case-specific experts, Drs. Weisbord and Sica. Both experts have explicitly stated that they accept the NSF diagnosis and will not dispute the role of the

September 2, 2005 Omniscan exposure had in causing it. And, in its trial brief, GEHC expressly states that it does "not dispute that NSF has created some limitations on Mr. Decker's ability to perform his normal activities of daily living." (Doc #: 149, at 4.)

Nonetheless, GE *argues* that Plaintiffs' motion on Mr. Decker's NSF diagnosis must be denied because evidence about his diagnosis is relevant to several important issues at trial "and would *permit a jury to find that Mr. Decker has another condition*." (Opp. Br. at 3 (emphasis added).) Moreover, "the jury may find that Mr. Decker has *some other condition* instead of or in addition to NSF." (Id. at 4 (emphasis added).) And, "Mr. Decker's clinical presentation *does not correlate well with a diagnosis of NSF*, leading the pathologist to question whether Mr. Decker has 'a deeper sclerosing process' that is not NSF." (Id. (emphasis added).)

It is clear that Plaintiffs have the burden of proving, by a preponderance of the evidence, that Mr. Decker has NSF – and if that issue is disputed, the Court cannot grant summary judgment on it. However, the Court has previously ruled that if GEHC disputes a plaintiff's NSF diagnosis, it must produce a case-specific expert who has examined the plaintiff and reached the conclusion that the plaintiff does not have NSF. If GEHC does not present this type of evidence, the Court will not allow it to argue that Mr. Decker does *not* have NSF.

With respect to causation, it is also Plaintiffs' burden to show that the single dose of Omniscan caused Mr. Decker's NSF. In its trial brief, GEHC does not dispute that Mr. Decker's September 2005 Omniscan-enhanced MRI, "along with other co-factors, was associated with his development of NSF." (Doc #: 149, at 4.)

While the Court will not grant summary judgment on specific causation, it will not allow GEHC to refute causation, for example, based on the Lemy study. In other words, unless GEHC

has an expert who has analyzed Mr. Decker and Patient 5 in the Lemy study and based upon this analysis and comparison concludes that Mr. Decker's NSF was not caused by his 2005 Omniscan.

IX.

On a related note, Plaintiffs ask the Court to exclude opinions relating to idiopathic or so-called gadolinium-naive NSF (**Doc #: 81**). The Court has reviewed the motion and the related briefs and concludes as follows.

There is no independent disease called "idiopathic NSF" or "gadolinium-naive NSF." There is one disease, and it is NSF. It is undisputed that at the very least there is a causal connection between GBCAs and NSF.

The Lemy study is a retrospective study of six kidney transplant recipients at a single institution during an 8-year period who were diagnosed with NSF. Five of the patients contracted NSF after exposure to a GBCA. One of the six (Patient 5) seems to have contracted NSF without a known GBCA exposure. As noted above, this fact is not relevant in the Decker trial unless GEHC has an expert who has analyzed both Mr. Decker and Patient 5 in the Lemy study and has determined that the conditions of the two of them are so similar that it is probable that Mr. Decker's NSF was not caused by his 2005 Omniscan.

X.

GEHC asks the Court to exclude expert testimony that "free" gadolinium caused Paul Decker's NSF. (**Doc #: 91**.) According to GE, because Mr. Decker has chosen not to have his tissue tested for the presence of gadolinium, he cannot legitimately say that he has gadolinium, free or otherwise, in him – or that free gadolinium caused his NSF. Nor should Plaintiffs'

experts be allowed to testify that free gadolinium is a general cause of NSF on the facts of this case. Absent evidence that Mr. Decker has gadolinium in his body, Plaintiffs and their experts must rely on general epidemiological evidence that, at best, suggests an association between GBCAs and NSF. Because the epidemiologic evidence does not identify an association between free gadolinium (as opposed to GBCAs) and NSF, Plaintiffs and their experts can only speculate that free gadolinium is a specific or general cause of Mr. Decker's NSF.

In response, Plaintiffs contend that GEHC's motion is a thinly-veiled attempt to exclude expert testimony that this Court has already found admissible concerning Omniscan's propensity to release and deposit gadolinium – the virtually universally acknowledged mechanism by which Omniscan causes NSF – and a concern of which GEHC was aware as far back as the 1980's. In other words, GEHC seeks to strike any evidence that the likely cause of Mr. Decker's NSF was a result of the release of gadolinium from the unstable Omniscan module.

This Court need not repeat its detailed reasoning, best articulated in Daubert I, for denying GEHC's motion to exclude Plaintiffs' generic experts from opining that de-chelated, or free, gadolinium causes NSF. (See Daubert I at 5-16.) In 2009, the Court summarized why Plaintiffs' free gadolinium theory passes Daubert and Rule 702 muster:

> The free gadolinium theory passes the relevancy test because it has a tendency to make the existence of any fact that is of consequence to the determination of the action more probable or less probable than it would be without that evidence. FED. R. EVID. 401. The free gadolinium theory passes reliability muster under Daubert because it is based on research conducted by scientists and doctors performing animal studies, *in vitro* studies, *in vivo* studies, human clinical studies and retrospective case studies along with review of the relevant published scientific and medical studies; the theory has been subjected to publication and peer review; the theory has been generally accepted in the relevant scientific and medical community; the Plaintiffs' experts have adequately accounted for obvious alternative explanations; and the research of Plaintiffs' experts relates not only to their review of the literature but to matters growing

> naturally or necessarily out of research they have conducted independent of this litigation. See Daubert, 509 U.S. at 593-94; FED. R. EVID. 702 Advisory Committee's Notes (2000 Amends.)
>
> Given the relevance and reliability of this theory, GEHC's challenge to that theory, which goes to weight of the evidence, is more properly made during cross-examination at trial rather than as a Daubert challenge to admissibility.

(Daubert I, at 11.)

It appears that the passage of time since the Court issued Daubert I has not diminished that theory, but strengthened it. Among other things, the 2012 American College of Radiology Manual states that "it is now generally accepted that GBCA exposure is a *necessary* factor in the development of NSF." (Doc #: 111-7, at 66 (emphasis added).) Furthermore, "[i]f the *prevailing hypothesis* is true – that the development of NSF is related to *the release of gadolinium from the chelates* that constitute GBCAs – the differences in number of reported cases may, in part, be explained by differences in chemical properties of different GBCAs."[6] (Id. (emphasis added))

The only competing causation theory of which this Court is aware is that of GEHC's expert and employee, Dr. Newton, who opines that chelated gadolinium (i.e., GBCA) is less stable than previously thought, and it is more likely that chelated (versus free) gadolinium triggers, at a cellular level, the fibrotic process leading to NSF. The Court questions how this theory helps GEHC, as GBCAs are chelated gadolinium. But in any event, GEHC is entitled to

[6]While the precise mechanism is unknown, there are a few reasonable theories related to characteristics of the gadolinium ion that are commonly considered to cause NSF (e.g., transmetalation, stimulation of microphages and macrophages leading to fibrotic cells present in NSF).

present its chelated gadolinium theory/defense, as Plaintiffs are entitled to present their free gadolinium theory.

For these reasons, the Court **DENIES** GEHC's motion (**Doc #: 91**).

IT IS SO ORDERED.

/s/ Dan A. Polster February 13, 2013
Dan Aaron Polster
United States District Judge